GENETIC AND CYTOLOGICAL STUDIES OF MAIZE

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The mode of action of controlling elements in maize, a main topic of recent reports, has continued to be studied during the past year. Attention has been directed particularly to the system of elements of which *Spm* (Suppressor-mutator) is a component. This system, which affects the action of genic substances at the A_1 locus in chromosome 3, has been described in previous reports and is referred to as the a_1^{m-1}-*Spm* system. It has been concluded that a_1^{m-1} arose by insertion of an element, belonging to the system of which *Spm* is a component, at the standard A_1 locus. This element directly affects the action of the gene substance at A_1, altering it in a recognizable way, and the modified A_1 locus has been designated a_1^{m-1}. It responds in readily detectable ways to the presence of the independently located element *Spm*, and one of the responses effects changes in gene action in both somatic and germinal cells. The *Spm* element may undergo change in location within the chromosome complement by a process termed transposition. Methods of detecting transpositions of controlling elements have been discussed in recent publications. One method takes advantage of the linkage relation between the element and a given genetic marker; transposition of the element to a new location alters this relation, and such alterations are easily detected. Two tests utilizing this method were conducted with *Spm* during the year, to reveal more about the degree of stability of its location. They will be summarized below.

The tests determined the numbers of *Spm* elements in different parts of individual plants, and their locations in the chromosome complement. In each test, the examined individuals represented the progeny of a single plant that carried one *Spm* element at a known location. It was possible to gain some information about the frequency of occurrence of transposition of *Spm* and the period during development when it takes place; but in these respects the results of the two tests were quite different. One gave evidence of frequent transposition of *Spm*, occurring early in development. The other showed infrequent transposition, and thus indicated a considerable degree of stability of location. Although transposition of *Spm* appears to be under genetic control, the factors and conditions associated with it have not yet been recognized.

The first test involved progeny of a plant in which *Spm* was carried in chromosome 9. This plant was a_1^{m-1}/a_1 (chromosome 3), *Wx*/*wx* (chromosome 9); and it had one *Spm* element in chromosome 9, as the testcross results entered in table 7, A, indicate. Twelve variegated plants, derived from variegated kernels in the *Wx* class of the first ear of this plant (see table 7, A, row 1, column 5), were tested for *Spm* number and location. The silks of all fertile ears produced by each plant received pollen from plants that were homozygous for a_1^{m-1} and *wx* and had no *Spm*.

Table 6 records the number of ears obtained from each plant and their positions on the plant, the *Spm* constitutions of the cells that produced these ears, and the linkage relations of *Spm* to *Wx*. All together, twenty-six ears were obtained from these 12 plants. In 1 plant, the cells that gave rise to the ear on a tiller (side branch) had no *Spm*, but in the cells that gave rise to the remaining twenty-five ears one or two *Spm* elements were present. In sixteen of

the ears, one *Spm* was present and was linked with *Wx*. In five ears, two *Spm* elements were present and one of them was linked with *Wx*. In four ears, one *Spm* was present, but it was not linked with *Wx*. The kernel types appearing on these three classes of ears are entered in B of table 7. It should be noted that the cells which produced the first and second ears on the main stalk, in the 5 plants from which such ears were obtained, had the same *Spm* constitution. In 3 of the 7 plants from which tiller ears were obtained, however, a difference was expressed between the cells of the main stalk and those of a tiller with respect to *Spm* constitution and location.

The results indicate that in the plants of this culture the *Spm* element underwent frequent change of location in the chromosome complement. The time of change was either late in the development of the germinal cells in the parent plant or early in development in the progeny plants. The transposition mechanism will account not only for the changes in location of *Spm* but also for the observed losses or increases in number of this element in different plants and in different parts of the same plant.

The second test of stability of location of *Spm* was made with the progeny of a plant carrying an *Spm* element located close to *Y* in chromosome 6. This particular location of *Spm* was detected initially only in one plant of a culture. That plant was a_1^{m-1}/a_1^{m-1}, *Y*/*y* in constitution; and the silks of one of its ears received pollen from a plant that was homozygous for a_1^{m-1} and *y* but had no *Spm*. The resulting ear was sectorial, in that a small sector at its base was composed of 47 kernels (21 Y:26 *y*) derived from cells in which *Spm* was absent. The cells producing the larger part of the ear carried one *Spm* element. Among the 329 kernels in this part of the ear, 167 had no *Spm;* 10 of them were *Y* and 157 were *y*. The remaining 162 kernels carried *Spm;* 153 were Y and 9 were *y*. Close linkage of *Spm* with *Y* was evident, for only 5.6 per cent recombinants appeared among the 329 kernels.

All fertile ears produced by 17 plants derived from the *Y, Spm* class of kernels

TABLE 6. *Spm* Constitution and Location in Different Plants of a Culture and in Different Parts of Individual Plants

Plant No. in Culture 7285	No. of Ears Tested per Plant	Position of Ear in Plant	*Spm* Constitution and Linkage with *Wx*
A-6, B-1, and B-6	1	1st ear, main stalk	1 *Spm;* linked with *Wx*
B-4	1	1st ear, main stalk	2 *Spm;* one linked with *Wx*
A-5	2	1st and 2nd ears, main stalk	2 *Spm;* one linked with *Wx* (both ears)
B-2 and B-5	2	1st ear, main stalk; tiller ear	1 *Spm;* linked with *Wx* (both ears)
A-1	3	1st and 2nd ears, main stalk	1 *Spm;* linked with *Wx*
		Tiller ear	1 *Spm;* not linked with *Wx*
A-3	3	1st and 2nd ears, main stalk	2 *Spm;* one linked with *Wx*
		Tiller ear	1 *Spm;* linked with *Wx*
A-4	3	1st and 2nd ears, main stalk	1 *Spm;* not linked with *Wx*
		Tiller ear	(all three ears)
A-2	3	1st ear, main stalk	1 *Spm;* linked with *Wx*
		Ear on one tiller	1 *Spm;* linked with *Wx*
		Ear on another tiller	No *Spm*
A-7	4	1st and 2nd ears, main stalk; ear on each of two tillers	1 *Spm;* linked with *Wx* (all four ears)

TABLE 7. Phenotypes of Kernels on Two Ears of 1 Plant (A), and on Twenty-Five Ears Produced by 12 Plants in Its Progeny (B)

Kernels in A derived from cross of ♀ a_1^{m-1}/a_1, Wx/wx × ♂ a_1^{m-1}/a_1^{m-1}, wx/wx, no *Spm*; in B, from cross of ♀ a_1^{m-1}/a_1^{m-1} or a_1^{m-1}/a_1, Wx/wx × ♂ a_1^{m-1}/a_1^{m-1}, wx/wx, no *Spm*.

No. and Location of *Spm* in ♀ Parent	Phenotype of Kernel						Total No. of Kernels
	Deep Color (germinal mutation)		Pale Color (no *Spm*)		Colorless with Spots of A_1 (*Spm* present)		
	Wx	*wx*	*Wx*	*wx*	*Wx*	*wx*	
A							
1 *Spm*; linked with *Wx*	0	1	26	196	197	18	438
	0	0	38	81	89	29	237
B							
1 *Spm*; linked with *Wx*	1	0	418	1539	1512	356	3826 *
2 *Spm*; one linked with *Wx*	0	0	79	267	594	323	1263
1 *Spm*; not linked with *Wx*	0	0	190	168	140	174	672

* 20.2 per cent are "recombinants."

received pollen from plants that were homozygous for a_1^{m-1} and *y* but had no *Spm*. One ear per plant was obtained from 3 plants, two ears per plant were obtained from 4 plants, three ears per plant from 7 plants, and four ears per plant from 3 plants. All together, forty-four ears were produced by these 17 plants, and in all of them the cells that gave rise to the ear carried one *Spm*. In forty-three of these ears, close linkage of *Spm* with Y was expressed; only in the ear produced by a tiller of one plant was there no evidence of linkage of *Spm* with Y. Table 8 records the phenotypes of the kernels that appeared on the forty-four ears.

In contrast to the first-described test, this test revealed a case in which the *Spm* element showed a considerable degree of stability of location. As was mentioned

TABLE 8. Phenotypes of Kernels Appearing on Forty-Four Ears Produced by 17 Plants Having the Constitution a_1^{m-1}/a_1^{m-1}; Wx/wx When Pollinized by Plants Homozygous for a_1^{m-1} and *wx* and Having no *Spm*

A. Phenotypes appearing on the forty-one ears produced by 15 of the plants. B. Phenotypes appearing on a partially sterile ear of one plant. Reduction in transmission of the Y-carrying chromosome is evident. C. Phenotypes appearing on the two ears produced by one plant; (1) main ear, (2) tiller ear.

Ear	Phenotype of Kernel						Total
	A_1 (germinal mutation)		Pale Color (no *Spm*)		Colorless with Spots of A_1 (*Spm* present)		
	Y	*y*	Y	*y*	Y	*y*	
A	2	2	247	4708	4551	192	9702 *
B	0	0	1	91	20	1	113
C(1)	0	0	25	203	171	11	410
C(2)	0	0	65	47	48	59	219

* 4.5 per cent are "recombinants."

earlier, however, nothing is yet known about the genetic or other factors that may control the time during development of a tissue when changes in location of *Spm* will occur, or the frequency of such changes.

Types of Spm Elements

The observed effects of *Spm* control over gene action at a_1^{m-1} have been remarkably consistent, notwithstanding the various different locations in the chromosome complement the element is known to occupy. Sometimes, however, the action of *Spm* becomes altered, producing modified types of control of a_1^{m-1} expression. One modification, which arises rather frequently, will be considered here. Occasionally there will appear, on an ear of a plant carrying a_1^{m-1} and *Spm*, a kernel with an aberrant phenotype. Instead of many deeply pigmented spots in a colorless background, this kernel may have only one or several tiny dots of deep pigmentation in a colorless background. Plants were grown from several kernels of this type, and they and their progeny were examined to determine the nature of the change responsible for the altered phenotype. Tests showed that in such cases an *Spm* element is present, but its capacity to suppress gene action at a_1^{m-1} and to induce mutation at that locus is much weakened. It has therefore been given the symbol *Spm-w*. In this part of the discussion, the standard *Spm* element will be designated *Spm-s* to distinguish it from the *Spm-w* element.

Spm-w elements have been found in several different chromosomes of the complement. The one to be considered here first appeared in a single kernel on an ear produced by a plant carrying *Spm-s* in chromosome 5. This kernel showed only a few dots of deep pigment in a colorless background. The plant that developed from it was pigmented throughout, in contrast to plants carrying *Spm-s*, which have streaks of deep pigmentation in a nonpigmented background. Tests revealed the presence in this plant of an *Spm-w* element, located in chromosome 5, which showed the same value of recombination with *Pr* as the *Spm-s* element in the parent plant.

Part of the progeny of the plant was examined, in turn, and tests of the different types of individuals it contained made it possible to define the weakened action of the *Spm-w* element. The reduced capacity of *Spm-w* to suppress gene action at a_1^{m-1} is shown by the appearance of anthocyanin pigment throughout plants that carry it. The development of this pigment, however, is very much retarded in comparison with that in plants that have no *Spm* element. Plants with *Spm-w* become fully colored only at late maturity, whereas plants with no *Spm* develop pigment at early stages. The weakened capacity of the *Spm-w* element to induce mutation at a_1^{m-1} is also shown by the phenotypes of kernels that carry it. Either mutant spots are totally absent, or just one or a few spots appear.

Table 9 has been constructed to show the linkage of this *Spm-w* element with *Pr*. When both *Spm-s* and *Spm-w* are present in the same plant, *Spm-s* is epistatic to *Spm-w*. Both elements segregate normally at meiosis, however, as illustrated by the kernel types on the ears of the testcross recorded in table 10. Each of the 5 plants used in this testcross carried *Spm-s* in chromosome 6, closely linked with *y*, and *Spm-w* in chromosome 5, linked with *Pr*.

Another type of *Spm* element has received preliminary examination, but description will be postponed until more is known about its action. *Spm* elements with altered expressions may represent changes in state of the standard *Spm-s* element. If so, their origins should be comparable to the changes in state of *Ac*.

A Modifier Element within the Spm System

An element which greatly increases the rate of mutation at a_1^{m-1} in the presence of *Spm* first appeared in a kernel on one ear of a plant that was a_1^{m-1} Sh_2/a_1 *sh*

TABLE 9. Phenotypes of Kernels Appearing on Ears Produced by 20 Plants from the Cross ♀ a_1^{m-1}/a_1^{m-1}; *Pr Spm-w/pr* × ♂ a_1^{m-1}/a_1^{m-1}; *pr/pr;* no *Spm-w*

Phenotype of Kernel with Regard to a_1^{m-1} Action	*Pr*	*pr*	Total	Per Cent Recombinants
Pale color (no *Spm-w*)	1016	2943	3959	25.6
Colorless with one or several A_1 dots or specks (*Spm-w* present)	1780	569	2349	24.2
Colorless; no A_1 specks (*Spm-w* present)	....	....	1462	....
Total number of kernels			7770	
Total with *Spm-w*			3811	

in constitution and carried one *Spm* element. The silks of two ears of this plant received pollen from a plant that was homozygous for a_1 and sh_2 and had no *Spm*. These two ears produced a total of 738 kernels; 354 had a_1^{m-1} and Sh_2, and 384 were homozygous for a_1 and sh_2. Of the kernels in the a_1^{m-1} Sh_2 class, 167 were uniformly pale colored (no *Spm*) and 187 had spots of deep color in a colorless background (*Spm* present). All but one of the variegated kernels exhibited the number of mutant spots that is characteristically produced by the state of a_1^{m-1} known to be present in the pistillate parent. One kernel, however, had a much larger number of mutant spots; and the plant grown from it showed a marked increase in number of streaks of deep pigmentation in a non-pigmented background. To investigate the reason for this altered expression of a_1^{m-1}, the plant was self-pollinated, was crossed reciprocally with a plant that had another state of a_1^{m-1} but no *Spm*, and was used as a pollen parent in crosses with plants that were homozygous for a_1 and sh_2 and had no *Spm*. The tests revealed the presence in this plant of an unmodified *Spm* element, and also an unmodified a_1^{m-1} locus. It had an independently located element, however, which was capable of markedly increasing the frequency of occurrence of mutation at a_1^{m-1} when *Spm* was also present; and both states of a_1^{m-1} responded to it in this manner. Plants were grown from the various classes of kernels appearing on each of the ears produced by the above-indicated crosses; and they, in

TABLE 10. Phenotypes of Kernels Appearing on Ears of 5 Plants Produced by Cross of ♀ a_1^{m-1}/a_1^{m-1}; *Y/y Spm-s* (standard *Spm*); *Pr Spm-w/pr* × ♂ a_1^{m-1}/a_1^{m-1}; *y/y; pr/pr;* no *Spm-s;* no *Spm-w*

Phenotype of Kernel with Regard to a_1^{m-1} Action	*Y Pr*	*Y pr*	*y Pr*	*y pr*	Totals
Pale color (no *Spm-s,* no *Spm-w*)	98	200	9	12	319
Many A_1 spots in colorless background (*Spm-s* present)	16	19	360	342	737
One or several A_1 dots or specks in colorless background (*Spm-w,* no *Spm-s*)	212	69	11	9	301
Colorless; no A_1 dots or specks (*Spm-w,* no *Spm-s*)	56 *Y*		5 *y*		61
Total kernels					1418

Summaries: 737 *Spm-s* (376 *Pr* : 361 *pr;* 35 *Y* : 702 *y*)
362 *Spm-w;* no *Spm-s* (223 *Pr* : 78 *pr;* 337 *Y* : 25 *y*)
319 no *Spm-s,* no *Spm-w* (107 *Pr* : 212 *pr;* 298 *Y* : 21 *y*)
29.8% recombination between *Pr* and *Spm-w*
5.7% recombination between *y* and *Spm-s*

turn, were tested for presence and absence of the modifier element, and for its inheritance behavior when present. These tests not only confirmed the conclusions drawn from tests of the parent regarding the presence and mode of action of the modifier element; they also showed that the element could undergo change of location in the chromosome complement in somatic cells. In this respect, therefore, its behavior is much like that of some of the other controlling elements.

The origin of this new controlling element, in the system of which the *Spm* element and the element at a_1^{m-1} are components, is not understood. It is known, however, that without the new element the types of mutation, their time of occurrence during development of a tissue, and their frequency of occurrence are expressions of the state of a_1^{m-1} itself. The modifier element affects the expression of only one of these three aspects of state, namely, the frequency of occurrence of mutation. When it is present in conjunction with one of the states, the increase in mutation frequency is estimated to be about threefold.

The Relation between a_1^{m-1} and a_2^{m-1}

It has recently been determined that the system of elements responsible for control of gene action at a_1^{m-1} also operates to control gene action at a_2^{m-1}. In this respect, the history of origin of both a_1^{m-1} and a_2^{m-1} is of considerable significance, and so will be outlined briefly.

Some years ago, in the progeny derived from self-pollination of a plant, a number of individuals exhibited variegation in leaf and sheath with regard to intensity of chlorophyll pigmentation. A study was commenced to examine the expression of this variegation and also its inheritance pattern. In the course of study, many plants in one culture that carried the control system regulating chlorophyll expression were self-pollinated. On the ear produced by one of these plants, some kernels exhibited variegation for anthocyanin pigmentation. Plants derived from the variegated kernels also showed variegation for anthocyanin pigmentation, and tests conducted with them made it possible to associate this phenotypic expression with an alteration that had occurred at the standard A_2 locus in one chromosome 5 of the parent. The modified locus was designated a_2^{m-1}, for in kernels carrying it spots of anthocyanin appeared in a colorless background, as if the change in gene expression was from recessive to higher alleles of A_2.

Study was continued, and in its course the silks of an ear of a plant carrying the system responsible for control of mutations at a_2^{m-1} received pollen from a plant that was homozygous for a_1, carried in chromosome 3, and for the standard A_2 locus, carried in chromosome 5. On the resulting ear, one exceptional kernel, instead of being totally pigmented, had spots of deep pigmentation in a colorless background. The plant derived from this kernel also exhibited variegation for anthocyanin pigmentation. Tests of the plant indicated the presence of the a_1 locus derived from the male parent and of an altered A_1 locus derived from the female parent. Alteration of the A_1 locus must have occurred late in development of the ear of the pistillate parent plant, for only this one kernel exhibited modified A_1 action. The modified locus was designated a_1^{m-1}. All studies of a_1^{m-1} have been made with progeny of this single plant.

Although similarities in expression were noted between the chlorophyll variegation originally studied and the variegations associated with the modified A_2 and A_1 loci (a_2^{m-1} and a_1^{m-1}), investigation of the operation of the systems responsible for control of gene action in the first two cases was suspended. Attention was concentrated instead on examination of the system responsible for control of gene action at a_1^{m-1}. When the mode of operation of that system, the *Spm*-a_1^{m-1} system, was appreciated, it became clear that further consideration should be given to a_2^{m-1}, to

determine whether or not changes in expression of genic materials at this locus are under the control of elements belonging to the same system. Pedigree relationships as well as kinds of behavior suggested such a possibility. Study of the system operating at a_2^{m-1} was therefore resumed, with this viewpoint in mind.

Two strikingly different states of a_2^{m-1} were selected for the renewed investigation. Both states respond to an independently located element that exhibits a Suppressor-mutator (*Spm*) type of control of gene action. With one of these states, some gene action occurs at the a_2^{m-1} locus in the absence of *Spm*, resulting in the appearance of anthocyanin in both kernel and plant, the pigment being uniformly distributed within a tissue. The rate of action appears to be lower than that of the standard A_2 gene, for in both plant and kernel the pigment intensity is low. In the presence of the *Spm* element, however, gene action is suppressed except in some cells where mutations at the a_2^{m-1} locus, initiated by *Spm*, allow the gene substance to be fully active. These mutations result in stability of expression of the genic materials at the locus in subsequent cell and plant generations. The characteristics of this state of a_2^{m-1} are essentially similar to those of some states of a_1^{m-1}.

The expression of the second selected state of a_2^{m-1} has not yet been satisfactorily analyzed, but it clearly differs from all the many known states of a_1^{m-1}. In the absence of the *Spm* element, it gives rise to deeply pigmented kernels and plants, although the intensity of color is not so great as that produced by the standard A_2 locus. When the *Spm* element is present, both kernel and plant are variegated. Pigmented areas in the kernel have about the same intensity as that produced in the absence of *Spm;* but colorless areas may be present within the pigmented areas. The pattern of variegation in any one kernel depends upon the number of *Spm* elements present. When only one *Spm* element is present, the kernels have many large pigmented areas as well as some small pigmented spots. When two *Spm* elements are present, there are few if any large pigmented areas, and most of the kernels have only small pigmented spots. If three or more *Spm* elements are present, the kernels either are totally colorless or have small pigmented spots in a restricted region of the aleurone layer. The type of pigment in the colored areas, and the observed effects of dose of *Spm* on the pattern of their appearance, suggest that the variegation in this case is not related to mutation at the a_2^{m-1} locus, as it is with the previously described state of the locus. Rather, the pigmented areas seem to reflect changes affecting the *Spm* element, which either result in its removal or alter its capacity to suppress gene action with this state of a_2^{m-1}.

The *Spm* element that was present in the cultures having the two states of a_2^{m-1} is not the same as the *Spm-s* element carried in the a_1^{m-1} cultures. Changes in its mode of control of gene action at a_2^{m-1} sometimes occur in somatic cells, and these are often expressed in different sectors of the same plant. They affect both the suppressor and the mutator action of the *Spm* element. The nature of these changes is not yet understood; but the marked difference in stability of action between this *Spm* element and the *Spm-s* element was strikingly revealed when the latter was introduced into an a_2^{m-1} culture. The *Spm-s* element exerts a consistent and stable control of gene action at a_2^{m-1}, of a type similar to that which it exerts at a_1^{m-1}.

On the other hand, the mode of inheritance of the *Spm* element in the a_2^{m-1} cultures is similar to that in the a_1^{m-1} cultures. Somatically occurring transpositions bring about loss of *Spm* from some cells, change of its location in others, or changes in both number and location. The several methods adopted to detect such transpositions in the a_1^{m-1} cultures have also been applied here. In addition, use of the state of a_2^{m-1}

that reflects doses of *Spm* has made it possible to select among the kernels on an ear those that have different numbers of *Spm* elements. The effectiveness of this selective method was shown by tests of 12 plants (group A) derived from kernels whose variegation patterns indicated the presence of more than one *Spm* element, and 10 plants (group B) derived from kernels whose variegation patterns indicated the presence of only one *Spm* element. One of the plants in group A had no *Spm*, which suggested that transposition, occurring in the gametophyte, had resulted in increase in number of the *Spm* element in the endosperm nucleus and its absence in the zygote nucleus. In tests of the remaining 11 plants, one testcross ear per plant was obtained from 5 plants, two testcross ears per plant from 5 others, and three testcross ears from the eleventh. In 1 plant, three or four *Spm* elements were present in the cells that gave rise to an ear on the main stalk and one on a tiller. Seven plants had two *Spm* elements in the cells that produced the main and the tiller ears. In the remaining 3 plants, two *Spm* elements were present in the cells that gave rise to the main ear, but only one *Spm* element in the cells that gave rise to a tiller ear.

Among the 10 plants in group B, one testcross ear per plant was obtained from 2 plants, two testcross ears per plant from 5 plants, three testcross ears from 1 plant, and four testcross ears per plant from the remaining 2 plants. All the ears, except two tiller ears of 2 plants, were produced from cells having one *Spm*. The cells that produced each of these tiller ears had two independently located *Spm* elements.

The finding that the same system of elements controls gene action at a_1^{m-1} and at a_2^{m-1} is not unexpected. Similar relationships with respect to control of gene action have been observed at a number of different loci in cultures carrying the *Ds-Ac* system. Insertions of the *Ds* element at different gene loci have initiated control of action of the genic substance at each locus by this system of elements. The origin of a_1^{m-1} by modification of a standard A_1 locus in a culture carrying the elements responsible for control of gene action at a_2^{m-1} suggests that at both loci an element of common ancestry is present. That element may also have been present at the locus of the gene responsible for chlorophyll variegation, for the chlorophyll variegate was the first-recognized member of this sequence of gene change. The possibility cannot be tested, however, since the cultures carrying it were discarded some years ago.

It is also not unexpected to find differences in expression of the *Spm* element in the a_1^{m-1} and a_2^{m-1} cultures, for a series of selections was made among the a_2^{m-1} cultures before the system responsible for control of a_2^{m-1} action was recognized. We know, too, that changes may occur in the action of the *Spm* element in a_1^{m-1} cultures, as described previously. Moreover, other elements may appear, such as the modifier of rate of mutation at a_1^{m-1} discussed above; and these, if their presence is not recognized initially, may be responsible for unwitting bias in the selection of kernels and plants for subsequent study. In this connection, it is suspected, although not yet certain, that an inhibitor of *Spm* may be present in some of the a_2^{m-1} cultures.

Obviously, since the number of variables may increase during the course of a study, analyses of systems of controlling elements can sometimes be complicated and time consuming. Recognition of the different elements belonging to a control system, and of the changes that may occur in them as regards both type of action and location in the chromosome complement, requires many types of test. In order to study any one element of a system, each of the other variables, as it is recognized, must be removed by crossing and selection, so as to work with the smallest possible number of associated and interacting elements.

Aberrant Behavior of a Fragment Chromosome

Much effort has been expended during the past year in analysis of a structurally modified chromosome 9, of which preliminary investigations were reported in Year Book 55. In this modification the substance of chromosome 9 is distributed between two components. The distal third of the short arm comprises one member, referred to as the fragment chromosome. At the proximal end of this segment is a centromere, from which may extend a small, deeply staining piece of chromatin. The extension is often lost, however, leaving the fragment with a terminal centromere. The other component carries the proximal two-thirds of the short arm and all of its long arm, and is referred to as the deficient chromosome. The fragment chromosome shows aberrant behavior in somatic cells. It may be lost to some cells, and undergo changes in structural organization in others. It may also become attached to ends or centromeres of other chromosomes, or be incorporated into another chromosome. Both the frequency of occurrence of events leading to such consequences and the time of their occurrence during the development of a tissue are now known to be under genetic control.

Interest in this structurally modified chromosome was aroused initially by the aberrant behavior of the fragment chromosome in somatic cells. Later it was discovered that this fragment could behave unexpectedly in some of the meiotic cells also, and in plants either heterozygous or homozygous for the structural modification. Although there is no conspicuous cytological evidence of the fact, it has been shown genetically that a segment of chromatin, adjacent to the centromere in the fragment chromosome, duplicates a segment at the end of the deficient chromosome. Products that could be assumed to arise from crossing over between this segment in the fragment and the homologous segment in the normal or in the deficient chromosome have appeared in the progeny of both heterozygote and homozygote. It is certain, however, that not all of them result from the ordered process of meiotic events that normally leads to crossing over; and this is made particularly evident in the apparent crossover products formed at meiosis in the homozygote. Often these are structurally normal chromosomes 9, but sometimes they are defective. The frequency of appearance of crossover products in the gametes of the homozygote varies widely among the different homozygotes, but is constant for any one of them. The present evidence, though limited, does suggest that the variation may be an expression of the genetic system that controls the time of occurrence of aberrant events altering the organization of the fragment chromosome. If so, then when this system operates in a meiotic cell the crossover mechanism may be utilized but the fragment chromosome itself may not be required to undergo the usual preliminaries that normally control the position of crossing over and its frequency of occurrence at any one position. At any rate, it is certain that the rules assumed to apply to crossing over in maize are not always followed by the fragment chromosome when it participates in a crossover type of event.

Until more definite conclusions can be drawn regarding the mechanism responsible for the complicated behavior of the fragment chromosome, a review of the evidence obtained from the many tests conducted with it will be postponed.

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Lahr, Ernest L., Associate in Research
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*Ozeki, Haruo, Research Assistant
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Wassermann, Felix, Guest Investigator
White, Harry, Chief Mechanic
Wilson, Carole E., Technical Assistant
Yoshida, Yoko, Research Assistant

Summer 1957 and Temporary

Anderson, Richard P., Maintenance Man
Baer, Harold, Guest Investigator
Beckwith, Barbara, Assistant
Bert, Grace R., Assistant
Burtch, Ethel P. (Mrs.), Typist
Cannon, W. Dilworth, Jr., Assistant
Fochtmann, Grace M., Assistant
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Gregory, Jack, Assistant
Holden, Floyd, Maintenance Man
Nevole, Nancy A., Assistant
Page, Gilbert, Maintenance Man
Powell, Florence (Mrs.), Assistant
Simrell, Elizabeth Jane, Assistant to Librarian
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Streisinger, Lotte, Assistant
Victoria, William, Maintenance Man

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Bernheimer, Alan W., Summer Investigator
Calef, E., Summer Investigator
Caspari, E. W., Summer Investigator
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Errera, M., Summer Investigator
Franzese, Eleanor, Business Manager
Granick, S., Summer Investigator
Hotchkiss, Rollin D., Summer Investigator
Hyde, Olive, Administrative Assistant
King, James C., Geneticist
Luria, S. E., Summer Investigator
Maramorosch, Karl, Summer Investigator
Novick, A., Summer Investigator
Skaar, Palmer D., Bacterial Geneticist
Wallace, Bruce, Assistant Director, Geneticist
Watanabe, T., Summer Investigator
Witkin, Evelyn M., Summer Investigator, Instructor

* Resigned during the year.